Gut, 1981, **22**, 759–762

Case reports

Treatment of chronic erosive gastritis with prednisolone

M J G FARTHING, P D FAIRCLOUGH,* J E HEGARTY, E T SWARBRICK,† AND A M DAWSON

From the Department of Gastroenterology, St Bartholomew's Hospital, London

SUMMARY Short-term oral prednisolone was successfully used in the treatment of four patients with chronic erosive gastritis which had failed to respond to conventional therapies. Symptomatic improvement occurred in all patients during the first week of treatment. Gastroduodenoscopy after two weeks showed that the erosions had healed. Remission has continued in these four patients for two, seven, eight, and nine months without maintenance therapy. This limited uncontrolled study suggests the corticosteroids may be of benefit in patients with chronic erosive gastritis who fail to respond to conventional treatment.

Chronic erosive gastritis (CEG) is characterised by a wide spectrum of symptoms extending from nausea and vague abdominal discomfort[1] to anorexia, vomiting, and gross weight loss mimicking a gastric carcinoma.[2 3] The condition is of unknown aetiology and tends to respond unpredictably to medical treatment.[4] The radiological and endoscopic appearances are distinctive[3–6] and consist of many mucosal bulges with central superficial ulceration, localised to the gastric antrum or extending diffusely from the distal oesophagus to the first part of the duodenum.

Symptoms are often not relieved by antacids and anticholinergics, although an uncontrolled study of the H_2 receptor antagonist, cimetidine, suggested that it may be of some benefit.[3] Gastric acid secretion and serum gastrin are normal, but histologically there is a marked increase in immunocytes, particularly IgE cells, which clearly differentiates this condition from chronic atrophic gastritis.[3] These findings suggest that allergic mechanisms may be involved and disodium cromoglycate, in a limited uncontrolled trial, was reported to help a small group of patients.[7] However, it is our experience that these remedies do not reliably induce remission, particularly when symptoms are severe. This observation prompted a limited uncontrolled study of corticosteroid therapy.

Methods

PATIENTS

Four consecutive female patients, 56–77 years old, took part in the study, two of whom had radiological and endoscopic evidence of chronic erosive gastritis for at least six months before entry. Two others had had symptoms for several months but the diagnosis had not been confirmed until entry into this study. All patients had failed to respond to conventional therapy, including antacids, cimetidine, and disodium cromoglycate. Double contrast barium meal and gastroduodenoscopy were performed during the week before starting corticosteroid therapy. Treatment consisted of prednisolone 40 mg daily orally for 10–14 days, after which endoscopy was performed by two observers, each of whom made an independent assessment of the severity and extent of the lesions. The dose of prednisolone was then progressively decreased during the next two to four weeks, after which it was discontinued and further endoscopy was performed. At each attendance patients' symptoms were assessed.

*Address for correspondence: P D Fairclough, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE.

†Present address: New Cross Hospital, Wolverhampton, West Midlands.

Received for publication 1 April 1981

Case 1
This 52 year old woman presented with a six month history of anorexia, vomiting, and 13 kg weight loss. Serum albumin was reduced at 32 g/l, but full blood count and ESR were normal. Barium meal examination and gastroscopy showed a high lesser curve gastric ulcer. Symptoms improved with bed rest and Caved-S but anorexia persisted. One year later on developing abdominal pain after meals which was not relieved by antacids, she was found to have the classic appearances of chronic erosive gastritis both radiologically and at gastroscopy. Again serum albumin was reduced at 30 g/l but other biochemical and haematological investigations were normal. During the next three months she remained in bed in hospital and received carbenoxolone 300 mg daily on which her symptoms improved and she gained weight. Endoscopy three months later showed a knobbly, irregular mucosa but only a few erosions. During the next three years the same symptoms returned intermittently and another admission was eventually precipitated by persistent vomiting and a weight loss of 19 kg. Gastroscopy showed extensive chronic erosive gastritis involving the antrum and body of the stomach and deep mucosal biopsy showed a dense chronic inflammatory cell infiltrate in the lamina propria with some polymorphs consistent with the diagnosis of chronic erosive gastritis. After seven days treatment with oral prednisolone the vomiting ceased, appetite improved markedly, and she gained weight. After another seven days, gastroscopy showed a red, irregular mucosa but no erosions were seen. Albumin rose from 32 to 37 g/l and gastroscopy six weeks after the start of therapy confirmed that the lesions had not returned. Double contrast barium meal examination, six months later, showed that radiological remission had been maintained. This patient has continued in clinical remission for nine months since completing treatment with prednisolone.

Case 2
A 55 year old woman gave a three month history of abrupt onset of anorexia associated with epigastric fullness and discomfort. She had not lost weight and routine haematological and biochemical investigations were normal. Double contrast barium meal examination showed extensive chronic erosive gastritis involving the antrum and distal body of the stomach, findings which were confirmed endoscopically. Symptoms persisted despite antacid therapy and during the next three months she lost 3 kg in weight. She was treated with disodium cromoglycate for six months (800 mg daily) with minimal symptomatic and no radiological improvement. At the end of this period of treatment she had lost another 2·4 kg in weight. After a gastroscopy, which showed that the appearances in the stomach had not changed during the past eight months, she was treated with prednisolone. Within two weeks there was marked symptomatic improvement, weight gain, and complete resolution of the erosions endoscopically, although the mucosa remained irregular with scattered 0·5 cm nodules. After another two weeks gastroscopy was repeated and a few erosions were seen on the lesser curve. A barium meal at three months showed that a few erosions persisted on the lesser curve but clinical remission has been maintained for eight months.

Case 3
A 74 year old woman gave a 10 year history of aching epigastric discomfort. Gastroscopy showed an irregular knobbly gastric mucosa but no ulceration was reported. During the next two years her symptoms were unchanged but a double contrast barium meal showed chronic erosive gastritis involving the antrum and the duodenal cap. After cimetidine (1 g daily) for four months her symptoms were moderately improved but were still present eight months later when radiological and endoscopic assessment was unchanged. Gastric biopsies showed infiltration of the lamina propria with lymphocytes and plasma cells which was consistent with the diagnosis of chronic erosive gastritis. Symptoms improved after two weeks' treatment with prednisolone and the erosive lesions regressed markedly, although gastroscopy four weeks later showed a small acute benign gastric ulcer (0·5 cm in diameter) on the lesser curve, which was confirmed histologically. Symptomatic remission has been maintained during the seven months' follow-up but she declined further radiological or endoscopic assessment.

Case 4
A 76 year old woman presented with an eight month history of abdominal swelling. There were no abnormal clinical findings and investigations, which did not include barium studies. were normal. Two years later she returned with profound anorexia, epigastric pain, and outpatient records confirmed that she had lost 11 kg in weight during the intervening two years. A presumptive clinical diagnosis of gastric carcinoma was made. As an interim symptomatic treatment she was given cimetidine which failed to improve her symptoms. Subsequently gastroscopy and barium meal demonstrated chronic erosive gastritis involving both the antrum and the body of the stomach. In view of the severity of her symptoms she was treated with prednisolone, which improved her symptoms dramatically within seven

days. After two weeks' treatment the lesions had healed, although the mucosa remained red and irregular. Six weeks after the initial gastroscopy, when treatment had been discontinued, two or three erosions were seen on the lesser curve. Remission of symptoms has been maintained for two months since completing treatment with prednisolone.

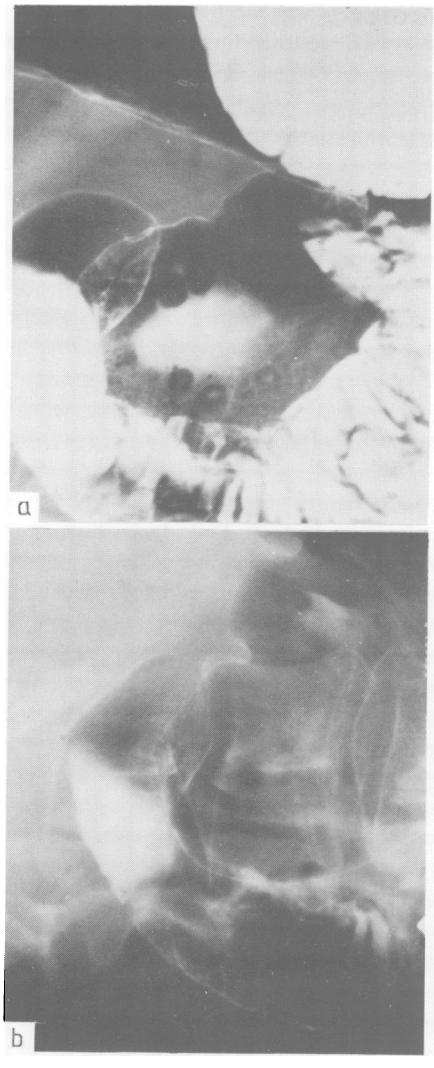

Figure *Case 2. Representative radiographs from double contrast barium meal examinations. (a) Classic lesions of chronic erosive gastritis in the gastric antrum. (b) Complete resolution after treatment with oral prednisolone.*

Results

Symptoms improved in all patients and this improvement has been maintained for up to nine months, which is the longest period of follow-up. The endoscopic and radiological findings confirm that the erosions regress rapidly after starting treatment with prednisolone, with marked improvement within two weeks. There were no adverse effects, although one patient (case 3) developed a small benign gastric ulcer during prednisolone therapy, although the erosions regressed. All patients are at present in remission on no treatment. The Figure (a) shows the classic radiological appearances of chronic erosive gastritis in the antrum of case 2, and complete resolution of the lesions three months after treatment with prednisolone (Figure, (b)).

Discussion

Chronic erosive gastritis is a relatively uncommon condition but the radiological and endoscopic appearances are distinctive[3] and differentiate it from the flat erosions of acute erosive gastritis[8] and the attenuated gastric rugae so typical of chronic atrophic gastritis and gastric atrophy in which it is unusual to find a breach in the mucosa.[9] Histologically, chronic erosive gastritis is distinguished from chronic atrophic gastritis by the elongation of gastric pits and the striking increase in IgE producing cells in the former.[3]

Chronic erosive gastritis is subject to cyclical relapses and remissions,[3] features which make interpretation of uncontrolled studies of therapy difficult. This limited uncontrolled study in four patients, however, strongly suggests that oral prednisolone relieves symptoms and promotes rapid healing of the erosions. Although this improvement could have occurred by chance during a spontaneous remission, two of the four patients (cases 2 and 3) had good evidence of activity of the disease, which had failed to respond to conventional therapy for several months before starting prednisolone. We can find no other variables that could have promoted such a rapid response in all four of these patients. The longest remission is in case 1—it has now lasted for nine months—and, as yet, the others at two, seven, and eight months have not relapsed. These patients received intensive short-term therapy but the place of medium or long-term maintenance treatment needs to be established.

For patients who achieve a satisfactory clinical remission with symptomatic treatment alone, there is no indication to pursue an endoscopic or radiological remission with corticosteroids. However, this study suggests that prednisolone may have a

place in the management of patients who are severely ill with chronic erosive gastritis or who fail to respond to conventional therapy. A controlled trial is indicated to test these suggestions, although in view of the relative rarity of this condition it would need to be performed in a centre which has a large pool of suitable patients.[3]

MJGF is supported by a grant from the Wellcome Trust and PDF holds a Senior Wellcome Clinical Research Fellowship.

References

[1]Albot G, Leger L, Tricard A. Formations polypoides a sommet ombliqué de la muqueuse gastrique. *Presse Med* 1947; **55:** 190–1.

[2]Moutier F, Martin J. Deux cas de gastrite varioliforme. *Arch Mal Appar Dig* 1947; **36:** 155–60.

[3]Lambert R, André C, Moulinier B, Bugnon B. Diffuse varioliform gastritis. *Digestion* 1978; **17:** 159–67.

[4]Walk L. Erosive gastritis. Clinical review and analysis of twenty-seven cases. *Gastroenterologia* 1955; **84:** 87–98.

[5]Roesch W, Ottenjann R. Gastric erosions. *Endoscopy* 1970; **2:** 93–8.

[6]Laufer I. *Double contrast gastrointestinal radiology with endoscopic correlation.* Philadelphia, London, Toronto: Saunders, 1979: 168–70.

[7]André C, Mouliner B, Lambert R, Bugnon B. Gastritis varioliformis, allergy and disodium cromoglycate. *Lancet* 1976; **1:** 964–5.

[8]Jeffries GH. Gastritis. In: Sleisenger MH, Fordtran JS, eds. *Gastrointestinal disease.* Philadelphia, London, Toronto: Saunders, 1978: 734.

[9]Chatterjee D. Idiopathic chronic gastritis. *Surg Gynec Obst* 1976; **143:** 986–1000.